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16. ABSTRACT

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There is, however, no single, available data base for assessing exposure to lead in drinking water. In this article, we use a variety of data sources to develop a profile of lead levels in US public drinking water supplies, providing some upper and lower bound estimates of likely exposures as well as identifying some risk factors.

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Exposure to Lead in US Drinking Water*

Ronnie Levin
Office of Research and Development, US Environmental Protection Agency, 401 M Street - SW, Washington, DC 20460, USA

Michael R. Schock
Office of Research and Development, US Environmental Protection Agency, Risk Reduction Engineering Lab, Cincinnati, OH 45268, USA

Allan Marcus
Battelle, Statistics and Data Analysis Systems, 100 Park Drive, Suite 207, Research Triangle Park, NC 27709, USA

Abstract

In the US, lead occurs primarily as a corrosion by-product in public drinking water supplies. That is, its source is the corrosive action of the water upon the materials used in the water distribution system and private plumbing. Historically, drinking water has not been a major source of lead exposure for most Americans. However, as other sources of lead exposure continue to decline in this country, particularly decreased airborne lead from reduced use of leaded gasoline and decreased dietary lead, the relative contribution of drinking water as an exposure source has increased. This has occurred simultaneously with increasing evidence that lead's health effects occur at lower exposure levels, levels previously thought to be "safe".
There is, however, no single, available data base for assessing exposure to lead in drinking water. In this article, we use a variety of data sources to develop a profile of lead levels in US public drinking water supplies, providing some upper and lower bound estimates of likely exposures as well as identifying some risk factors.

Introduction

Lead is a metal with many commercial applications. Lead is so closely associated with the conveyance of water that the very word *plumbing* is derived from the Latin word for it. Lead is also used in manufacturing gasoline, paint,

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automobile batteries, glass and crystal, pencils, plastic pipes, pesticides, newsprint, cable sheathing and as a fruit growth regulator. Lead contaminates every environmental compartment (air, soil, water, *etc.*) and is a serious health and environmental problem in both industrialized and developing countries (Nriagu, 1988). Drinking water is a significant source of lead exposure in the United States and in the world.

Sources of Lead Exposure

In the United States, the general population is exposed to lead in air, food, soil and dust, in addition to water, which will be discussed here separately. The routes of exposure are inhalation and ingestion, but some occupational environments (such as the manufacture of tetraethyl and tetramethyl lead) may also present a risk of dermal absorption.

Sources of lead exposure other than drinking water

The US Environmental Protection Agency (EPA) monitors ambient air lead levels for compliance with the Clean Air Act. At current levels, relatively little lead is inhaled from ambient air, but ambient air contributes lead to dust (EPA, 1989). For children, the lead content of dusts can figure prominently in total lead exposure; EPA estimates that >40% of baseline (*i.e.* virtually unavoidable) lead exposure, excluding drinking water, results from ingestion (or, less often, inhalation) of dust (EPA, 1986a). Dust also accounts for >90% of the additive lead attributable to living in an urban environment or near a smelter (EPA, 1986a). Soil contaminated by lead, from lead particulates in the air or from the "chalking" or scraping of lead-based paint, can also contribute significantly to blood lead levels, particularly for children. Because there are no national estimates of soil lead levels in the US, the contribution of average soil-lead to average blood-lead has not yet been estimated reliably.

Average blood lead levels in the United States decreased significantly during the late 1970s and early 1980s. This decline in blood lead levels probably resulted primarily from the reduction in lead in gasoline, with a subsequent reduction in ambient air lead levels (Annest *et al.*, 1983), and from reductions in dietary exposure (EPA, 1985; EPA, 1986a).

Adults and older children in the United States now receive most of their baseline exposure from food ingestion (excluding drinking water and lead attributable to an urban environment). Lead may be added to food either from atmospheric sources or during food processing. Atmospheric lead may be added to food crops in the field or pasture, during transportation to the market, during processing, or during kitchen preparations. Processing of foods, especially canning, can significantly increase the lead content of foods. However, the use of lead solder in cans produced and filled domestically has decreased greatly in the past decade. Lead used in pottery glazes can also contaminate food in some circumstances, and isolated cases of resulting lead toxicity have been documented (*e.g.* Wallace *et al.*, 1985; Weisskopf, 1987).

Lead in drinking water

Lead can contaminate drinking water at two stages: at the water source or through the use of materials containing lead in the water distribution system. Relatively few US public water supplies have lead in the source water. In its recently proposed National Primary Drinking Water Regulations for Lead and Copper (Federal Register 53: 31516-31578), EPA estimated that about 1% of the public water systems (served by surface or groundwater or both) have water entering the distribution system with lead levels >5 $\mu g\ L^{-1}$. (These data are discussed under Data Sources, below.)

The principal source of lead in US drinking water is the corrosion of materials containing lead used in the water distribution system, particularly service lines and private plumbing. The highest concentrations of lead are found where pipes or solder containing lead are used in combination with corrosive waters (*i.e.* water that dissolves lead easily), where water has been sitting for many hours in pipes, or where there is newly-installed plumbing or repaired joints using flux or solder containing lead. Lead levels are dependent on many factors that are both building-specific (*e.g.* age of plumbing) and time- specific (*e.g.* stagnation period) which causes significant variability in lead levels between and even within water systems.

Risk Factors for Elevated Lead Levels in Drinking Water

The principal sources of lead in drinking water are lead interior or service line plumbing and components; lead:tin soldered joints in copper plumbing; and brass and bronze valves and fixtures. Until the recent ban on the use of materials containing lead in public water supplies and in residences connected to them (Safe Drinking Water Act as amended in 1986, Section 1417), most soldered joints in copper plumbing were made with solder containing at least 40% lead. Common brass and bronze alloys used in the drawing or casting of faucets and valves contain approximately 2-8% lead. (The allowable limit is 8% lead.)

The impact the Ban will have is hard to predict. The legislation was passed in 1986 but was not incumbent upon the States until June 1988. Certainly, the ban on the future use of the fairly- ubiquitous lead solder to join copper pipes and the less common but continued use of lead pipes and fittings will slowly but significantly reduce the highest risks associated with the exposure. Several major uncertainties about the future impact remain: the continued contribution of existing lead solder and pipes and brass and bronze faucets and components; impurities in copper pipes and plastic pipes; the contamination of water in drinking water fountains; factors related to lead levels in multistory buildings and, especially, the level of enforcement of the Ban itself. In addition, brass and bronze in the past have generally only contained up to 8% lead, so the Ban may not affect this exposure source. Finally, do-it-yourself-ers are unlikely to be affected by this legislation. EPA's Inspector General is currently investigating the enforcement of the Ban.

Chemistry of lead solubility

Through the oxidation of the elemental lead constituting the pipe, lead is corroded and can then be available for uptake into the water. The oxidation process and the subsequent dissolution and transport of lead is enhanced by high oxidant levels (*e.g.* high dissolved oxygen, high chlorine dosages), low pH, and by the complexation of free lead ion (Pb^{2+}) by ligands such as hydroxide, carbonate, ammonia and polyphosphates. Lead corrosion is limited by the formation of "passivating" films on the surface of the pipe, usually of relatively insoluble corrosion products of lead. The level of soluble lead that will result from dissolution is a function of the concentrations of several important constituents in the water that participate directly or indirectly in film formation. Important protective deposits in simple water chemistry systems are the basic lead carbonates $Pb_3(CO_3)_2(OH)_2$ (hydrocerussite) and $Pb_{10}(CO_3)_6(OH)_6O$ (plumbonacrite). Orthophosphate and silicate lead solids also are generally even more protective, but their exact forms have not yet been determined very well. Solids such as lead oxide (PbO, massicot and litharge), lead carbonate ($PbCO_3$, cerussite), and lead (IV) oxide (PbO_2, plattnerite) have also been identified in coatings from some waters. Deposits of non-lead compounds that act as diffusion barriers to slow the migration of lead into the water could also reduce lead exposure, by slowing the rise of the water lead concentration.

Most of the chemical reactions responsible for the formation of passivation films or diffusion barriers on pipe are reversible in relatively short timeframes (days to months). This causes the production of "seasonal" trends in the data, or other changes in metal levels in response to chemistry changes. Inhibitors, because they rely on the formation of passivation films of lead corrosion products, must be constantly added above a threshold level for maintenance of passivation layers (Schock and Wagner, 1985; Schock, 1989b). Periodic changes in water chemistry, even at nearly identical sites, will cause variability in observed lead levels. Systems can undergo considerable variation in water quality resulting from intentional or unintentional excursions in water parameters, such as pH, alkalinity, chlorine, chloride, phosphate, *etc.*

The oxidation and corrosion of lead from soldered joints is more complicated than the processes involving pipes. Additional factors that are important are the direct chemical attack of solder by acidic or ammoniated flux that dissolves lead and spreads it into the pipe interior, and galvanic action between the pipe material (copper) and the solder in the joints (*e.g.* Lyon and Lenihan, 1977). Lead is present in the solder essentially as a diluent, and does not participate in the binding to the copper or alloying with the tin (Parent *et al.*, 1988). Oliphant (1983) found that lead solubility considerations do not give a particularly accurate estimate of the effect of various anions on lead uptake into the water from the galvanic corrosion normally displayed by soldered joints. Rather, the corrosion rate at a soldered joint is determined by the aggressiveness of the water supply (which is hard to quantify), plus the surface characteristics of the individual soldered joint.

There are several ways in which brass is attacked by drinking water.

Unfortunately, most studies of water chemistry effects have focused on "meringue" dezincification, or forms of material (or device) failure, and they did not directly measure metal leaching by different types of water (Schock and Neff, 1988). Lead is not alloyed with other metals in the brass and is just disseminated throughout the material. The distribution of lead is not necessarily purely random; it may be affected by the drawing or casting process and be rendered either more or less active from a corrosion and leaching standpoint. Water chemistry probably has some effect on the metal leaching extent and rate, but such studies have not been done in any suitably controlled fashion. No direct correlation between percentage of lead in the alloy and potential for water contamination has been demonstrated, although such a relationship cannot be ruled out.

The corrosion of any materials containing lead can generate both dissolved and particulate lead as contaminants. The particulate lead can result from the sloughing-off or dislodging of corrosion deposits on the pipe, joint or fixture surface. Different corrosion deposits have different abilities to withstand flow turbulence. Particulate lead or lead-containing material may also be produced by erosion of brass or exposed solder on the inside of pipe. Normal sampling protocols, which do not include filtration in order to better approximate exposure, will be influenced by the appearance of "spikes" of high lead concentrations, a major manifestation of the presence of particulate lead. Particles, if large enough, can become trapped by faucet aerators and contribute to prolonged elevated lead levels if the contact time is sufficient. Lead can also adsorb on other sediment in plumbing systems and be transported in that manner. Such sediment can result from plumbing repair, oxidized iron generated in the system, residual aluminum precipitating after pH change in the distribution system, *etc.*

Other risk factors for elevated lead levels in drinking water

The *age of the material containing lead* will have a different influence on the lead level at the tap, depending upon the nature of the material, the water or both. Lead pipe can continue to contribute lead indefinitely. That contribution is a function of the water chemistry and the nature of surface deposits, which can slow leaching rates or limit equilibrium lead solubility. Unless there are chemical changes over time, the deposits will yield a relatively constant amount of lead. The exception to this would be the exposure of new surfaces, where corrosion films must form and age.

On the other hand, lead leaching from soldered joints and brass faucets appears to decrease over time (Lee *et al.*, 1989; Schock and Neff, 1988; Sharrett *et al.*, 1982; Britton and Richards, 1981. Therefore new housing (under five years) or housing where plumbing renovation or repair has been done within the past few years, has a greater risk of producing high lead exposure than older housing (*e.g.* Sharrett *et al.*, 1982). Whether this observed phenomenon is the result of the formation of a passivation layer, or the result of physical depletion of lead through dissolution or erosion, is not known with certainty. Water chemical characteristics almost certainly affect the rate and duration of the lead

leaching. However, controlled studies have not been performed to quantify the significant factors. The Ban will reduce much of the highest risk associated with new housing.

The time the water spends standing in a pipe, when flow is shut off, is the *"stagnation" time*. Once stagnation commences, the lead pipe or surficial lead-containing deposit will begin to dissolve. The dissolved lead concentration will rise until it levels off at its equilibrium solubility value for those specific water chemistry conditions. The curve describing the rise in concentration has a characteristic shape, and has been described either from theoretical mass-transfer considerations (Kuch and Wagner, 1983) or purely by empirical statistical models (Bailey and Russell, 1981; Bailey *et al.*, 1986). The curves generally show a rapid initial increase within the first few hours of standing. The curves then level off gradually as the equilibrium solubility level is approached. Depending on water chemistry and pipe (or fitting) conditions, 8-24 hours or more could be necessary to reach equilibrium levels under laboratory conditions (Schock and Wagner, 1985). The presence of films on the surface can retard the dissolution and transfer process, making the initial limb of the curve more shallow and lengthening the time to equilibrium (Kuch and Wagner, 1983). The importance of the time factor is that lead levels increase quickly upon standing, and that relatively small differences in stagnation time in the first several hours can result in relatively large differences in observed lead levels (a factor of 2 or more) under otherwise identical conditions.

Temperature contributes to variations in lead chemistry and exposure potential in three ways. First, the solubility of lead corrosion scales and deposits changes with temperature. Though no comprehensive studies have thus far been performed to quantify the temperature coefficients of the solubility and aqueous equilibrium constants governing lead chemistry in potable waters, temperature effects have been noted (*e.g.* Taylor and Lopata, 1984). Different solids and different aqueous species respond to temperature changes to different degrees. Second, chemical reaction rates tend to increase as temperature increases, so lead levels will be relatively larger at stagnation times less than needed for equilibrium. Temperature differences of 10°C for short time periods (on the initial steeply-climbing limb of the stagnation curve) could easily produce a two-fold increase in the lead level for many locations (Schock *et al.*, 1988). Third, other chemical equilibria in a water system change with temperature, thus indirectly influencing the lead level. For example, warming a water that is kept out of contact with the atmosphere and which does not deposit calcium carbonate or hydroxide solids will result in a pH decrease (Schock, 1989b). The magnitude of the pH change will mostly depend on the initial pH and inorganic carbonate concentration of the water. Other weak acid/base systems can influence the change, as well as significant amounts of complexation of pH-determining species. Conversely, temperature decreases produce increases in pH under similar constraints. Much of the data reported in the literature for changes in lead concentration with temperature may actually represent the result of changes in pH or other significant chemical parameters (Schock, 1989a,b).

Therefore, without adequate knowledge of the background of the data, extrapolations to other systems and water qualities cannot be reliably made.

The fact that most tap water samples represent nonequilibrium conditions, coupled with the materials, chemistry, and physical effects just summarized, means that there is a high degree of *variability* in lead levels under "normal" conditions. An analysis of data from studies where an attempt was made to repetitively sample under similar stagnation conditions at the same sites over time showed that the lead monitoring data tended towards a log- normal distribution and had a variability approximately directly proportional to the mean concentration (Schock, 1989a; Schock *et al.*, 1988).

The *physical positioning* of the lead source and the *usage pattern* of the building also exert strong influences on the probability of exposure to lead. Obviously, residences or old buildings having lead interior plumbing as well as lead service lines produce the highest potential for risk. The *diameter* and *length of the pipe* are also important. Lead is picked up faster from pipes of smaller diameter, and for a given diameter, with longer travel through the pipe (Kuch and Wagner, 1983). High surface area to volume ratios, as are found in faucets or poorly-made soldered joints, also exacerbate the risk potential.

Homes or buildings with lead service lines or lead "pigtails" (the connection between the main and service line) can develop "plugs" of high lead concentration in water maintaining contact with those pipe segments. A similar situation on a smaller scale pertains to soldered joints in copper plumbing or brass fixtures. Water coolers or building chiller systems feeding bubblers can have lined storage tanks that may contain lead. Significant lead levels can also be produced in these devices or systems through recirculation and accumulation of sediment of soldering residue or sediment containing adsorbed lead.

The *pattern of water consumption* in any building will govern whether water is received that represents contact with these lead sources, and therefore, a higher risk. For a location having relatively new plumbing containing lead solder or brass or bronze components, the particular area of concern would be the first-draw sample after some period of stagnation that would catch water standing in the faucet and in contact with soldered joints. The *volume of the sample* will be important in determining the lead concentrations, as well as whether or not any water was "lost" before collection. The amount of water use elsewhere in the home or building system prior to or concurrent with consumption or sampling can influence the lead level, in a logical but unquantifiable manner. Commercial buildings, large apartment buildings and schools have the additional complexity that the systems are rarely completely "flushed".

The Nature and Significance of Drinking Water as a Source of Lead Exposure

Estimating lead levels in US drinking water

Estimating exposure patterns for lead in drinking water is difficult because there is no nationally representative data base on lead levels in drinking water.

Because lead occurs primarily as a corrosion by-product, levels are generally much higher in drinking water in residential or other buildings than in the water distribution system. Several national studies of drinking water quality have been conducted that focussed primarily on the quality of water leaving the distribution facility, not the lead levels likely to occur at the tap.

EPA's data base for compliance with the Safe Drinking Water Act, for instance, addresses drinking water typical of the distribution system and not tap water. According to EPA regulations, Maximum Contaminant Levels (MCLs) are defined as "the maximum permissible levels of a contaminant in water which is delivered to the free flowing outlet of the ultimate user of a public water system". (40 CFR section 141.1(a)) The States and the water utilities have generally understood "free flowing outlet" to refer to a "fully flushed sample". Also, the procedures for laboratories certified for reporting purposes under EPA's Laboratory Certification Program, administered by the Office of Drinking Water, specify that the sample be taken after running the water for two or three minutes (EPA, 1982). These procedures decrease the likelihood of detecting lead contamination of drinking water and, consequently, data for compliance purposes do not accurately represent exposure to lead in US drinking water. (New testing guidelines for schools developed in response to the Lead Contamination Control Act of 1987 do address lead occurrence as a corrosion by-product. But this guidance focusses primarily on water fountains in schools and will not likely affect residential exposure.) When EPA promulgates new regulations for lead in drinking water (now scheduled for late 1990), EPA will also likely develop new laboratory certification requirements to conform to the new regulations.

EPA's baseline assessment of environmental exposures to lead, presented in the *Air Quality Criteria (Document) for Lead* (EPA, 1986a), relied upon the Market Basket Survey, conducted by the (US) Food and Drug Administration, for estimates of water consumption, drinking water lead concentrations, and dietary lead levels. However, the Market Basket was not designed to capture exposure to lead in drinking water. For instance, few water samples are taken, and the water was flushed before the sample was taken. Consequently, dietary exposure resulting from lead contaminated drinking water was underestimated in both the Market Basket Survey and the *Criteria Document* analyses based upon it, and EPA's baseline assessment of lead exposure presented in the *Criteria Document* does not include some first-draw drinking water as an unavoidable source of lead. The next revision of the *Criteria Document* will review these issues.

Nor are there any nationally representative studies of lead levels in water fountains and coolers, in the workplace or in schools. Isolated anecdotal reports of highly contaminated drinking water in specific locales appear regularly throughout the country. As examples, these reports concern contamination in 1) *local schools* in Boston (reported in the Boston Globe beginning May, 1988 and continuing through September); in Broward County, Florida (reported in the Sun-Tattler on March 31, 1988); in Portsmouth, New Hampshire (reported in the

Portsmouth Herald on May 18, 1988, and continuing); and in the Washington, D.C. area (reported in the Washington Post most recently on March 24, 1989); 2) in *specific office buildings* (such as the USA Today building, reported in the Washington Post on March 1, 1989 and confirmed April 1, 1989); and 3) in *randomly selected multistory buildings* (such as in New York City, reported in the New York Times on November 17, 1988).

In addition, data are now being presented in the US that show evidence of individual cases of lead poisoning attributable to a drinking water source. For instance, both the New Hampshire and Massachusetts Departments of Health reported at the 1988 Conference of the New England Consortium of Childhood Lead Poisoning Programs cases of lead toxicity within the previous year that were determined to be attributable to drinking water; one case of a child in new housing was also described in Cosgrove *et al.*, 1989. Also, a well-publicized case of lead toxicity in Suffolk County, NY in 1984 (reported in Murrell, 1985, and elsewhere) provided the impetus for the 1986 Lead Ban amendment to the Safe Drinking Water Act. In the United Kingdom, drinking water has long been regarded as a major contributor to blood lead levels, including many cases of toxicity (*e.g.* Sherlock *et al.*, 1982; Elwood *et al.*, 1984; Richards and Moore, 1984). Also, recent data on specific instances of contamination caused by electric water coolers resulted in the passage of the Lead Contamination Control Act of 1988, which mandated the recall of many water coolers and also addressed school drinking water. While such stories appear regularly and confirm the significance of this exposure source, they do not constitute an adequate data base for assessing such exposures nationally.

On the other hand, with the exception of some well-publicized corrosion control efforts such as in Boston and Seattle, there is no information that average lead levels in drinking water have changed significantly in the past decade or two in the United States. Therefore drinking water probably contributes to the residual or intercept term in equations relating environmental lead exposures to human blood lead levels in studies that did not accurately characterize drinking water exposure. (In such equations, the intercept term accounts for exposures unexplained by other measured sources. This constant term is unaffected by variations in specific contributions from particular sources. As such, it represents that residual blood lead contribution not accounted for by the individual sources of lead exposure measured in the study.) In various studies of environmental exposures to lead, controlling for exposures from air, soil and dust, typical intercepts are in the blood lead range of about 3 $\mu g\ dL^{-1}$ (Yankel *et al.*, 1977) to about 8 $\mu g\ dL^{-1}$ (Angle and McIntire, 1979); lead in drinking water is likely to contribute to that residual.

The nature of drinking water as an exposure source

There is discussion in the literature about the extent of exposure to unflushed water. Data from the U.K.'s Water Research Centre (WRC), based upon a small sample of households from WRC staff, indicated that most of the water that is drunk is "random daytime draws" (84%), and that relatively little (4%) is "first-draw" (Bailey and Russell, 1982). They also estimated that with a "typical"

three-person household, the mean random daytime stagnation interval is as short as 30 minutes. However, in the United States >76% of the male population over 16 years old and >55% of women over 16 are employed, and almost 60% of US children have mothers who work full or part-time (Statistical Abstracts, 1988); it is unlikely that the mean daytime stagnation interval in this country is 30 minutes. In addition to increasing the average stagnation interval, this also implies that for many US households, there are two "first-flush" conditions per dwelling unit per weekday: one in the morning and the other upon return from work. Furthermore, the WRC model does not consider that lead levels are tap specific, *e.g.* that running the bathroom faucet does not necessarily reduce first-draw lead levels in the kitchen. The WRC merely ascertained how often *any* water was used in the home. Data on the effect of stagnation interval upon lead levels indicates that there may be two or more "first-flushes" per day at each tap, *i.e.* in the bathroom and in the kitchen.

The relationship between water-lead and blood-lead levels

There are many studies relating blood lead to water lead (*e.g.* summaries in EPA, 1986a; EPA, 1986b). The relationship has either been characterized as linear or non-linear, most often a cube root function. EPA has re-analyzed much of the data from these studies, especially those evaluating water lead levels in the range typical of the US, *i.e.* <50 $\mu g\ L^{-1}$.

The blood lead *versus* water lead slopes can be calculated for this range even if the relationship described in these studies is non-linear, *e.g.* cube root. Whenever the raw data were presented in a tabular or graphical form in the published report, or could be obtained from the investigators, EPA reanalyzed the data with a logarithmic transformation of the linear relationship.

$$\ln(PbB) = \ln(a + b\ PbW).$$

This nonlinear expression of a linear model allows an estimate of the geometric mean blood lead concentration (PbB) for each water lead concentration (PbW), which is believed to be more appropriate for the highly skewed distributions typical of PbB and PbW, and indeed of interindividual variability in general (Hattis *et al.*, 1987; *etc.*).

In studies where multiple water samples were collected, standing samples related as well or better to blood lead levels than running samples in children (*e.g.* Worth *et al.*, 1981) and adults (*e.g.* Moore *et al.*, 1977; Pocock *et al.*, 1983; Moore *et al.*, 1982; Worth *et al.*, 1981). (For a discussion of sampling methods, *cf* Britton and Richards, 1981.) The only better measures have been the composite water samples collected from kettles in use at the time, which directly measure consumed water (Lacey *et al.*, 1985), and for homebound infants and their mothers, random daytime grab samples (Central Directorate, 1985). The data relating blood lead levels to lead levels in running samples are not inconsistent with those for standing levels: where comparable models are given or can be derived, the slopes for the running water lead studies are larger than those for standing levels. These results are summarized in Tables 11-51 and 11-52 of EPA, 1986a.

Table 1 *First-flush water lead-blood lead slopes for range 10-25 μg L^{-1}.*

Publication	Subjects/analysis	Slope (b)	Model
Worth *et al.* (1981)	524 Boston subjects	0.05	non-linear
	EPA reanalysis	0.04	linear
Moore *et al.* (1979)	232 Glasgow mothers	0.14	cube root
Hubermont *et al.* (1978)	70 pregnant Belgian women	0.05	linear
UK Central Directorate (1982)	28 Glasgow mothers	0.09	cube root
		0.009	linear
Lacey *et al.* (1985)	reanalysis	0.062	linear
	EPA reanalysis	0.05	linear
UK Central Directorate (1982)	126 Glasgow infants	0.12	cube root
		0.018	linear
Lacey *et al.* (1985)	reanalysis	0.07	linear
	EPA reanalysis	0.062	linear
Pocock *et al.* (1983)	7735 middle-aged men	0.062	linear
Moore (1984)	568 Scottish mothers	0.13	cube root

Source: Adapted from EPA, 1986a; Table 11-51 for 10-25 μg L^{-1} range.

For convenience, in Tables 1 and 2 we present the estimated blood lead *versus* water lead slopes from a variety of studies, in μg dL^{-1} blood lead per μg L^{-1} water lead for the interval 10-25 μg L^{-1} water lead. (These calculations are based upon Tables 11-51 and 11-52 of EPA, 1986a.)

The linear first-draw slopes re-estimated by EPA in Table 1 are reasonably consistent for adults (0.04, 0.05, 0.05, 0.062). The most reliable first-draw slope estimate for infants is larger, about 0.062. The linear running-water slopes in Table 2 are also reasonably consistent across adult studies (0.043, 0.072, 0.067, 0.07). The typical adult first-draw slope is about 0.05, while the typical adult running-water slope is somewhat larger, about 0.07. The running-water slope for the Glasgow infants is about 0.07, not much larger than the EPA re-estimated first-draw slope of 0.062 (Lacey *et al.*, 1985). However, we have recently reanalyzed the composite water lead data, taking into account that the blood lead levels at composite water lead <15 μg L^{-1} are much lower than expected. The linear relationship over most of the range 10 to 25 μg L^{-1} is much flatter, with a slope of about 0.047 (Marcus, 1989c). Thus the ratio of blood lead slopes for running water lead *versus* first-draw water lead is about 0.07/0.05 = 1.4 for adults, across the several studies.

Table 2 *Running water lead-blood lead slopes for range 10-25 μg L^{-1}.*

Publication	Subjects/analysis	Slope (b)	Model
Worth *et al.* (1981)	524 Boston subjects	-	-
	EPA analysis	0.0425	non-linear
Worth *et al.* (1981)	390 adult subjects	-	-
	EPA analysis	0.073	linear
Worth *et al.* (1981)	249 adult females	-	-
	EPA analysis	0.067	linear
UK Central Directorate (1982)	128 Glasgow mothers	0.10	cube root
		0.014	linear
	EPA reanalysis	0.07	linear
UK Central Directorate (1982)	126 Glasgow infants	0.11	cube root
		0.033	linear
	EPA reanalysis	0.07	linear
Sherlock *et al.* (1984)	114 adult women	0.14	cube root

Source: Adapted from EPA, 1986a; Table 11-52 for 10-25 μg L^{-1}.

There are several possible explanations why blood lead levels may so closely correlate with lead levels in standing samples. First, undoubtedly, people are exposed to some unflushed water. The WRC estimate of 4% may be a reasonable lower bound for such exposure. Second, several studies indicate that gut absorption of lead is much higher when ingested on an empty stomach (35% absorption *versus* 10%, Rabinowitz *et al.*, 1980) or between meals (Chamberlain *et al.*, 1978). The most likely times for exposure to elevated lead levels in drinking water coincide with these vulnerable periods: in the early morning, after school or work, and upon retiring. As a consequence, a relatively small fraction of consumed water may have a disproportionate influence on total lead absorbed. Another explanation relates to the nature of statistical analyses. Most exposure assessments use regression or correlation analysis to evaluate the relationship between changes in one variable (for instance, air lead) and changes in blood lead levels. Flushed water has much less variability than standing water; because there is less variability in the water measure, it can explain little variability in blood lead levels. This does not mean that the two variables are unrelated - just that little of the variability in the dependent variable is related to changes in the independent variable.

Estimating the contribution of drinking water to average blood lead levels

Estimating the relative contribution of drinking water to average blood lead levels has been difficult. Local studies in Boston (Worth *et al.*, 1981), Glasgow (Lacey *et al.*, 1985), Birmingham (UK DOE, 1982; Davies *et al.*, 1987) and Edinburgh (Raab *et al.*, 1987) show that, while there is significant scatter in the

results, drinking water appears to be a major source of environmental lead exposure there; however, these are all areas known to have unusually high water lead levels. On the other hand, lead levels in other studies are low, often as an artifact of sampling method. In Rabinowitz *et al.* (1985), for instance, where homes were visited at different times of the day, the samples were flushed to standardize the water collection protocol and to ensure comparability of measurements; however, this artifactually reduced the lead concentrations and eliminated most of the variability in the drinking water measurements, resulting in a variable with little predictive power. Nonetheless, even with this downward bias, the tap water lead levels in the high- lead group were significantly higher than in the lower lead groups. A national exposure profile cannot be generated from these studies.

Another complication in compiling a nationally representative assessment of lead exposure from drinking water is that, while it seems that the *absolute* levels of lead in drinking water have not changed significantly in the past 10 or 20 years, because other sources have declined dramatically, the *relative* contribution from drinking water has increased.

EPA attempted to estimate the average impact of drinking water as a source of lead exposure in support of the 1985 proposal to reduce lead in drinking water (EPA, 1986b). That study used data collected in 1979-1981 on lead levels in partly-flushed water from kitchen taps throughout the country. Those data led to an estimate that about 42 million of the 219 million people served by public water systems receive water with >20 $\mu g\ L^{-1}$ at the tap, and that average lead levels in partly flushed water at kitchen taps were about 15 $\mu g\ L^{-1}$, or slightly lower. EPA presented both this analysis and additional data in the most recent drinking water proposal.

A recent article (Bois *et al.*, 1989) attempted to use the pharmacokinetic concept of "clearance" to evaluate the blood lead-water lead relationship and the average contribution of water lead to total body lead burden. (The "clearance" concept assumes that for any compartmental pharmacokinetic model with constant rate coefficients and completely linear kinetics, the concentration of toxicant in a central pool, such as blood or plasma, will eventually approach an equilibrium level. At equilibrium, the quantity of toxicant "cleared" from the pool is exactly equal to the uptake of toxicant from the environment.) This concept generally has great utility, although some of the factors relating to lead concentrations in the body complicate its use.

One complication is that lead concentrations are partitioned in a complex and dynamic way among the blood (for recent exposures), the soft tissues, and bones (the site of long-term storage). The equilibrium half-life of lead in blood reflects a balance among lead in all tissues. A long-term equilibrium lead level is never achieved in adults (and probably not in children) due to the extremely long residence time of lead in cortical bone, at least 5-7 years (Schutz *et al.*, 1988). An approximate half-life between lead in blood and soft-tissues may exist, but this apparent half- life is known to increase with increasing bone lead burden (*e.g.* O'Flaherty *et al.*, 1982). Thus a non-equilibrium definition of clearance is

needed for lead.

Another problem with this analysis is that the clearance concept was used to assess the relative contributions of exposure from a single source (drinking water) but the baseline (blood lead levels) has changed by a factor of two over the time period discussed (*e.g.* Annest *et al.*, 1983, for US data). An exposure source that has remained constant in absolute terms will be a more significant relative contributor as other sources decline and total exposure decreases. In addition, sources of lead exposure are additive, suggesting that we should consider the absolute increment of lead burden assignable to drinking water, as well as the relative contribution (Houk *et al.*, 1989).

Another problem with this model is that the researchers used the same regression equation for children and for adults. Finally, the use of cross-sectional studies to evaluate the water lead- blood lead relationship with the clearance concept appears to have some methodological problems that have not yet been addressed.

We also attempted to estimate the average water lead contribution to average US blood lead levels (Marcus, 1989c). The piecewise linear blood lead *versus* water lead relationship derived in Marcus and Holtzman (1988) was integrated over an assumed log- normal distribution of water lead levels, whose parameters (geometric mean or median, and geometric standard deviation) were derived from the Patterson/Culligan data discussed below. If the distribution of Patterson/Culligan data are representative, then, using data to relate water lead levels to blood lead levels summarized in EPA, 1986b, about 2.4 $\mu g\ dL^{-1}$ is the typical average contribution from water lead to the blood lead of a young child. That average blood lead contribution of 2-3 $\mu g\ dL^{-1}$ was about 15% of the total exposure in the 1970s when average blood lead levels of young children were 12-15 $\mu g\ dL^{-1}$ (EPA, 1986a); it is projected to constitute about half of total exposure in 1990 (EPA, 1989). This analysis did not include exposure to lead in drinking water in the workplace, at school or from water fountains or coolers.

Data Sources

As discussed above, no single, available study of lead in drinking water is adequate to assess exposure to this source. However, some combinations of studies can provide upper or lower bounds of exposure, or can identify specific risk factors which can then be evaluated in terms of potential exposures.

Patterson study/Culligan data

One source of data is the widely cited study by Patterson Associates (1981), in which they analyzed 772 municipal water samples collected by Culligan Water Softener dealerships in 580 cities in 47 states. The samples were collected by a standardized protocol, after 30 seconds of flushing at a moderate flow rate at the kitchen tap, and stored in virgin polypropylene bottles. Samples were collected in May-November, 1978, mostly between 10 a.m. and 8 p.m.

There are some concerns with these data. For instance, the study population or sampling frame of the study was not defined in advance, but was drawn from

volunteer households with access to Culligan dealerships. This method of recruitment contains a self- selection bias, which cannot be estimated. While the sample proportions by state are roughly proportional to state populations, except for some over-representation of Illinois and under-representation of California, the representativeness of the data with respect to water treatment, plumbing system age, pH, and other factors is unknown. The data suggest that the waters were harder than average, possibly introducing a downward bias. In addition, the sampling protocol called for flushing the tap for 30 s before taking the random daytime sample; this protocol introduces elements of variability due to individual differences in performance and unknown stagnation periods.

It is not clear how accurately these data approximate the distribution of water lead levels in the US population. The Patterson study/Culligan data nonetheless suggest that a substantial number of US homes have a significant lead contamination problem, even in partly flushed water.

Marion County (Indianapolis, Indiana) data

In early 1987, the Marion County Health Department conducted a survey of drinking water lead levels in Indianapolis homes (Murray, 1987). Marion County water is extremely hard, with hardness ranging from 260 to 780 mg L^{-1} as calcium carbonate, and moderate pH (about 7). Samples were collected from 77 homes built within the previous 2-6 years and from 125 other homes, and the sampling protocols used were based upon the Patterson study/Culligan data discussed above: the sample was taken after flushing the kitchen tap for 30 s. A difference between the protocols was that in Marion County the water had sat for 8 h before being flushed for 30 s. In the Culligan/Patterson data the samples were random daytime draws, with an unknown sitting time.

Because of the hardness of the water, water softeners were used in 23% of the newer homes. PVC plastic pipes were present in 27% of those homes.

American Water Works Service Company

In January to March, 1988 the American Water Works Service Company (AWWSC) conducted a survey of the characteristics of the local water systems they manage/control, including analyzing trace element concentrations in tap water samples. They sampled 1484 sites, from 94 systems mostly in the Eastern and Midwestern US, at a sampling rate of 1:1000 accounts. The sites were randomly selected, with essentially proportional samples for each water supply district (system or part of a system), but with spatial clustering of sites permitted in some cases to allow more convenient sampling. The results are thus likely to be representative of the system from which they were taken. However, they may not be representative of the country. For instance, they are not geographically representative and there may be higher than average number of systems using corrosion inhibitors. We believe, however, that within strata for system characteristics such as inhibitor use, the samples are likely to be representative of within-system and between-system variation. A possible downward bias also may have been introduced because the samples were taken during the winter, which has been associated with lower lead levels in several studies.

The protocols called for sampling after the water had been sitting at least 6 h and up to 18 h. The first sample (bottle 1) included the first 100 mL from the faucet, and the second sample (bottle 2) took the next 1,000 mL from the tap. Bottle 3 was drawn after flushing the water for 2-3 min. Samples from sites with lead service lines were collected from a tap close to the service line, not necessarily at the kitchen faucet.

The original data set was reduced to 1,301 observations by removing cases with missing or out-of-range data on lead concentration, pH, water district or inhibitor use, and plumbing material. There were still missing values for other variables, such as plumbing age or presence of lead solder in the service line. We constructed a "one liter morning first draw" (MFD) data set from the AWWSC data by: (1) assigning the value 2.25 $\mu g\ L^{-1}$ to all nondetect samples from bottle 1 or bottle 2 (the detection limit was 5 $\mu g\ L^{-1}$); (2) calculating an MFD water lead concentration (W) from the bottle 1 and 2 concentrations PbW1, PbW2, weighted by sample volume

$$PbW = 0.1\ PbW1 + 0.9\ PbW2;$$

(3) if the calculated lead level was less than the detection limit, we called it a "non-detect" so as to better simulate our treatment of real MFD one-liter samples. The distribution of lead levels, for the whole sample and within strata, could always be well approximated by a log-normal distribution.

Massachusetts public schools data

In the summer of 1988, the Massachusetts Department of Public Health (MDPH) undertook a survey of lead in the drinking water of Massachusetts public schools to estimate the prevalence of elevated lead levels. This investigation followed a well- publicized study of elevated lead levels in Boston public schools done by a local television station. From 39 geographically stratified and randomly selected public schools throughout Massachusetts, 462 samples were collected and analyzed for lead and pH.

All samples were taken after an overnight stagnation period. At each school 12 samples were taken. Three samples were taken at the tap closest to the water meter; they included a first flush sample, one after the temperature change, and a fully flushed sample. Four samples were taken at two water bubblers or coolers in the building, each fountain supplying one first-flush and one 2-minute flush sample. Five samples were taken at sinks; two samples (both first-flush samples) were taken from sinks throughout the school and three samples (first-flush, 2-minute flush, and 10-minute flush) were taken from the sink most distant from the water meter. These samples probably represent the full range of exposures in these schools, and are likely to be fairly representative of exposures in schools in Massachusetts.

National Inorganics and Radionuclides Survey

The National Inorganics and Radionuclides Survey (NIRS) was conducted by EPA's Office of Drinking Water in 1984-1986 to collect information on the occurrence of selected inorganic compounds, including lead, and radionuclides

in finished drinking water from ground water sources throughout the country (EPA, 1986c). The NIRS sampled water from 1,000 randomly selected public water supplies with ground water sources and was designed to be representative of these systems. The quality assurance and quality control procedures were developed to ensure the representativeness of the data. Analyses were done in EPA labs.

Sampling procedures called for fully flushing the tap before the sample was taken. The data collected reflect levels in water typical of the distribution system and not typical of tap water lead levels.

Estimated Distribution of Water Lead Levels in the US

These data sets together provide some indications of likely contamination levels, or plausible upper- or lower-bounds for exposure, of lead levels in US drinking water. The three data sets for which there are an adequate number of samples for statistical analysis (Patterson/Culligan, AWWSC, and NIRS) indicate that lead levels tend to be log-normally distributed, although the variability in the measurements results in a "fat" tail. This permitted us to use basic statistical methods.

Lower bound of exposure

The NIRS data, which measured lead levels in water typical of the distribution system, provide a plausible lower-bound for baseline lead exposure from drinking water. In that survey, about 5% of the drinking water samples from fully-flushed taps exceeded 5 $\mu g\ L^{-1}$ (EPA, 1986c). Subsequently, all positive samples (*i.e.* those over the detection limit) were resampled. After resampling, 80% of the positive values fell to below detection limit. In its recent proposal to regulate lead and copper in drinking water EPA used the resampling data (*i.e.* 1%) as its estimate of lead levels >5 $\mu g\ L^{-1}$ in distribution water. However, given random measurement and analytical variability and the wide degree of variability typical of lead measurements in water (*e.g.* Schock *et al.*, 1988), resampling the high values only introduces a strong downward bias to the results. The 1% estimate probably underestimates this exposure source.

Therefore we conclude that between 1% and 5% of the US public water supplies deliver water with more than 5 $\mu g\ L^{-1}$. These data represent a lower-bound for exposure to lead in drinking water because some exposure to unflushed water is, as discussed above, probably unavoidable.

Identification of risk factors

Because it is the largest of the data sets available to us, and because there are ample data on water system and plumbing characteristics, the AWWSC data provide the best assessment of the contributions of various risk factors to lead levels in drinking water. The data are nearly log-normally distributed from 5 to >50 $\mu g\ L^{-1}$ for the whole data set and within virtually every stratification of the sample that we examined using log- normal cumulative probability plots. The mode or median of the log-normal distribution is best estimated by its geometric

Table 3 *Statistics of constructed one-liter first-draw samples from AWWSC data set, using the censored log-normal distribution.*

Average stratum	N	Percent NA	pH	ALK<30	Inhibitor	Statistic GM	Q80	Q90	Q95	GSD
						($\mu g\ L^{-1}$ water lead)				(No Units)
pH < 8	1,044	219	7.4	11%	33%	1.1 (0.141)	5.9 (0.085)	14.0 (0.081)	28.7 (0.108)	7.2 (0.112)*
pH ≥ 8	257	31	8.5	37%	7%	0.5 (0.452)	2.8 (0.245)	6.8 (0.197)	14.1 (0.220)	7.4 (0.308)*
Inhibitor	357	44	7.4	22%	---	0.5 (0.398)	2.8 (0.217)	7.1 (0.176)	15.3 (0.198)	8.3 (0.273)*
No inhibitor	944	206	7.7	14%	---	1.2 (0.141)	6.2 (0.087)	14.6 (0.090)	29.6 (0.112)	7.0 (0.113)*
ALK < 30	212	24	7.9	---	37%	0.6 (0.496)	2.8 (0.268)	6.3 (0.209)	12.6 (0.229)	6.6 (0.333)*
ALK ≥ 30	1,089	226	7.6	---	26%	1.1 (0.140)	5.8 (0.084)	13.8 (0.086)	28.5 (0.106)	7.3 (0.111)*
Solder	1,041	210	7.6	18%	31%	1.0 (0.156)	5.6 (0.092)	14.1 (0.094)	30.3 (0.115)	8.2 (0.122)*
No Solder	208	35	7.6	9%	10%	1.8 (0.220)	4.8 (0.124)	8.0 (0.116)	12.1 (0.140)	3.1 (0.164)*

* Asymptotic standard error of ln(statistic) shown in parentheses.
GSD: Estimated geometric standard deviation, dimensionless.
GM: Estimated geometric mean or 50th percentile.
NA: Number of observations in stratum above detection limit.
N: Total observations in stratum.
Q80, Q90, Q95: 80th, 90th, and 95th percentiles.

mean or GM = exp (mean of ln(Pb in water)), where exp () is the exponential function. The dispersion of this skewed distribution is best characterized by its geometric standard deviation or GSD = exp (standard deviation of ln(Pb in water)).

A problem in analyzing these data was that the majority of the samples were reported as "below detection" (the detection limit was 5 $\mu g\ L^{-1}$). However, because of the log-normality of the data, the geometric mean and geometric standard deviation can be estimated by extrapolating the straight line on a log-normal probability plot from the detected data, or rigorously using maximum likelihood estimation (MLE) methods. MLE can be calculated with a pocket calculator and auxiliary tables (Schmee *et al.*, 1985), or with microcomputer programs such as SYSTAT SURVIVAL. MLE methods are used here because they have known small-sample confidence intervals and facilitate estimation of log-normal percentiles, hypothesis tests for treatment effects, *etc.*, in spite of the left-censoring of the non-detect data.

Table 3 presents the AWWSC data set as reported in Marcus, 1989b and 1989d. The data were stratified by several of the system-wide factors believed to be most important in controlling tap water lead levels. These include: acidity (pH ≥8 *versus* pH <8); use of a system corrosion inhibitor such as zinc orthophosphate or sodium hexametaphosphate, *versus* no inhibitor use; total alkalinity (≤ 30 $mg\ L^{-1}$ *versus* >30 $mg\ L^{-1}$). The presence of lead solder is also an important factor, as these levels were sampled before full implementation of the 1986 lead ban. We have not included plumbing age in this table, but analyses using a subset of the data for which plumbing age was available showed that lead levels were always higher in buildings with young lead solder in the water distribution system (Marcus, 1989d). This difference persisted, even when the system water supply had high pH or had used a corrosion inhibitor.

Table 3 shows the number of buildings (N) in each pair of strata. The number of lead values above the detection limit (NA) is also shown, since the precision of the maximum likelihood estimates depends much more on NA than on N. The parameters estimated included the geometric mean (GM) of lead levels, which is also its 50th percentile. Since NA is less than 25% of N in all strata, this could never be verified from these data. We have thus also estimated the 80th, 90th, and 95th percentiles from the log-normal model (denoted Q80, Q90, Q95). The observed or empirical percentiles are in very good agreement with percentiles estimated from the log-normal model, increasing our confidence that the censored log-normal distribution (censored at the detection limit) is a good basis for estimating the effects of water characteristics on the distribution of household first-draw water lead levels.

An important role is played by pH, and the upper percentiles of the distribution are about twice as high in low pH water (<8) as in less acid water. The proportional variability, measured by GSD, is about the same. Systems which use corrosion inhibitors can expect to find percentiles that are at most half as large as those in non-inhibitor systems. Systems with higher total alkalinity may have higher percentile values than low-alkalinity systems, but more detailed

analyses show that this is attributable to the lower pH in these systems (Marcus, 1989c).

Lead solder is an extremely important factor. The observable percentiles (80th-95th) are much higher for units with soldered plumbing. Note that the fraction of houses below the detection limit is about the same in either case, about 80%. Above that limit, the corresponding percentiles become much higher for lead soldered plumbing. This is captured by the very large difference in variability, GSD = 8.19 for lead soldered plumbing and GSD = 3.13 for others.

We are also evaluating the role of repeat-sampling variability in lead levels in tap water.

Mid and upper range exposure estimates

Higher estimates of exposure are indicated by the Indianapolis data and the Patterson study/Culligan data. With the latter data set, as with the AWWSC data, the majority of observations (60%) were below the reported detection limit (10 $\mu g\ L^{-1}$). The "tail" in this data set was quite "fat", with 16% of the samples ≥20 $\mu g\ L^{-1}$ and 3% ≥50 $\mu g\ L^{-1}$. These data do not show the same highly significant correlation between pH and lead levels, although lead levels do decrease as pH increases in the pH range of drinking water (pH 7-10); minimum lead levels were associated with pH of 8. Interactive effects were indicated among key variables, including pH, bicarbonate ion, and calcium carbonate species.

The Indianapolis data show the bounds of a high-risk group within a relatively low-risk environment. In this study, 18% of the newer homes had lead levels >20 $\mu g\ L^{-1}$, as opposed to 6% of the older homes. Of the newer homes, 8% were >50 $\mu g\ L^{-1}$ while under 1% of the older homes were. Generally, overall lead levels in the older homes were lower, though age of house was not significant at the $p = 0.05$ level, and older homes with elevated lead levels tended to have lower levels than those in the newer homes.

While lead levels >20 $\mu g\ L^{-1}$ were much more common in Indianapolis homes with copper pipes than PVC pipes (27% *versus* 4%), it is noteworthy that one home with plastic pipes did have an elevated lead level. Early tests of plastic pipes showed that lead contamination resulting from stabilizers used with PVC pipes could be high, often >50 $\mu g\ L^{-1}$ (*cf.* summary of data in NAS, 1982; volume 4). However, since then the National Sanitation Foundation has developed a standardized testing procedure for plastic pipes that should have eliminated this problem.

In addition, again while not statistically significant at the $p = 0.05$ level in the Indianapolis study, lead levels in homes without water softeners tended to be higher than in those with softeners. The relationship between softener use and lead contamination levels is unclear and little information is available.

Finally, the data from Massachusetts show the highest lead concentrations. From school drinking water fountains, 42% of first-flush samples exceeded 20 $\mu g\ L^{-1}$, and 21% of these samples >50 $\mu g\ L^{-1}$. A two-minute flush reduced the MDPH data to 16% >20 $\mu g\ L^{-1}$ and 3% >50 $\mu g\ L^{-1}$. Because New England has long been known to have particularly corrosive waters (*e.g.* O'Brien, 1976; Karalekas *et al.*, 1978), these results should be understood as high exposures but

well within the range encountered by the general population. Schools with the highest risks (*i.e.* corrosive water and new plumbing) were not tested, so these data are not a high upper-bound. These school data may be typical of other non-residential exposures such as offices, factories, *etc.* served by corrosive waters. While drinking fountains are only a small source of exposure for most people, almost all water from them is consumed. For those with regular exposure to such sources, say in schools or for those in full-time work situations, water fountains may provide a significant source of lead exposure.

The MDPH data are not comparable to the Patterson/Culligan in that these are morning first-flush samples and two-minute flushes, while the latter are partly-flushed, random daytime grab samples. In addition, these samples were taken at schools and not in homes. But the data are consistent: the first-flush levels and the corrosive waters are associated with significantly higher lead concentrations. And they confirm the many local reports, cited throughout this article, of highly contaminated drinking water in particular locales or conditions.

A group with an identifiable risk of elevated water lead levels not fully included in these data sets are inhabitants of new homes. The Indianapolis data show a mid-range estimate of this risk. To assess the potential national risk associated with new housing, about 1.5-2.0 million housing units are built each year (Department of Commerce, 1988), with an average occupancy of 2.73 people per unit. Because lead levels can remain high for 2-5 years, this population is not insignificant at present. It is expected to decrease in the future as a result of the Safe Drinking Water Act ban on the use of materials containing lead in potable water systems. People living in existing housing but with recent plumbing repairs share this risk.

Conclusions

There is no single, available study of lead contamination of drinking water that adequately assesses this exposure. Combining available data sets, however, presents a qualitative profile of exposure to lead in drinking water: while most of the US population probably has relatively low lead levels, a substantial proportion has chronically high exposures. Furthermore, because of the great variability in lead concentrations, probably the majority of the population has sporadic exposures to high lead levels in drinking water, due to first-flush conditions, new plumbing, water coolers containing lead materials, use patterns in non-residential buildings, plumbing configurations in large or high-rise buildings, *etc.* Overt lead poisoning from tap water is uncommon but not unknown, and lead poisoning prevention programs in several states have begun to consider drinking water as a source of toxicity when other exposure sources are found to be low.

Elevated lead levels pose a public health problem with some unique aspects. The primary sources of lead in tap water are common and include lead-based solders, lead pipes and fittings, brass faucets and fittings, and components of water fountains that contain lead. Average lead burdens from drinking water are

moderate in size, typically 2-3 $\mu g\ dL^{-1}$ blood lead, but often much higher, resulting infrequently in cases of overt toxicity. The relative importance of drinking water as an exposure source is increasing in direct proportion to decreases in other sources. By the 1990s, EPA estimates that the contribution from drinking water will account for about half of average national exposures. In many cases the contribution from drinking water may be low to moderate, but the benefits of reducing such widespread exposures are large and estimatable.

Finally, this exposure is largely preventable. The easier part is to eliminate new sources of exposure, following the requirements of the Safe Drinking Water Act and the Lead Contamination Control Act. Reducing exposure from existing sources by reducing the corrosivity of water is also easy, and is actually economically beneficial for the longevity of public and private plumbing systems (EPA, 1986b). The relative costs and benefits of lead pipe replacement are unknown. Further research is needed in all of these areas.

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